Miscellaneous Causes of Pediatric Chest Pain

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There are many causes of chest pain, both cardiac and noncardiac, and as cardiovascular disease is the primary cause of death in the United States, physicians and parents are often appropriately concerned when a child presents with chest pain. Fortunately, cardiovascular disease is rarely the source of chest pain in the pediatric population. As one of the primary causes of sick visits to primary care and emergency physicians, pediatric chest pain may lead to pediatric cardiologist consultation.[1,2] This article describes some of the miscellaneous etiologies of pediatric chest pain that are important to recognize early and diagnose. Up to 45% of pediatric chest pain cases may elude definitive diagnosis, and these patients are often labeled as having idiopathic chest pain.[3] Serious morbidity or mortality is infrequent. Accurate diagnosis of more obscure causes may help to avoid unnecessary referral and can alleviate the concern and stress families and patients experience when dealing with chest pain.

PRECORDIAL CATCH SYNDROME

Precordial catch syndrome refers to a common cause of pediatric chest pain that was first described in 1955 by Miller and Texidor.[4] It is often referred to as Texidor twinge.[5] The pain is described as a sudden onset, sharp, stabbing, midsternal or precordial chest pain without radiation. Patients generally can point to the area of maximal pain, although the pain or tenderness is often absent at the time of evaluation, because the pain characteristically resolves without intervention. Typical duration of the pain is from 30 seconds to 3 minutes. The pain may be exacerbated by deep breathing, but it is not associated with dyspnea, cough, or other respiratory findings. The most common age of presentation for precordial catch syndrome is 6 to 12 years.

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The pathophysiology of precordial catch syndrome is unknown, although it is hypothesized to originate from the parietal pleura,[5] from rib cartilage, or the chest musculature and boney structures. Like most causes of chest pain, there is a broad differential when encountered in the pediatric patient. Actual diagnosis is made with a thorough history, lack of findings on physical examination, and normal ancillary testing, although electrocardiography and chest radiography are not indicated as part of the initial evaluation of precordial catch syndrome. If history and physical examination alone cannot rule out other causes of chest pain, such as spontaneous pneumothorax, rib fractures, or pericarditis, then ancillary tests may be necessary.[6]

Management and treatment of patients diagnosed with precordial catch syndrome is supportive. Family and patient anxiety may be high surrounding chest pain, particularly if there is a family history of cardiovascular disease. Reassurance is required. Analgesic medications may not be helpful, as the pain is self-limiting and of short duration.

CHEST PAIN ASSOCIATED WITH COCAINE OR METHAMPHETAMINE USE

Illicit drug use is frequent, with an estimated 19.9 million illicit drug users in 2007, which represented 8% of the population over 12 years of age. After marijuana, cocaine was the second most commonly used drug in the United States, accounting for 0.8% of the population over the age of 12, which equates to 2.1 million users. Overall, rates of illicit drug use among 12- to 17-year-olds have decreased in the United States, from 11.6% in 2001 to 9.5% of the population in 2007. A similar decrease was seen in 18- to 25-year-olds, although the use of prescription pain medications has recently been on the rise.[7]

Chest pain associated with both cocaine and methamphetamine use is described in the literature, and it is important for health care workers to remember that people often combine illicit drugs and concomitantly use tobacco, alcohol, or prescription medications. Chest pain associated with cocaine use is often described as tight rather than sharp, is typically confined to the chest and arm, and can be associated with diaphoresis, nausea, vomiting, or dyspnea. The incidence of these symptoms is similar to patients presenting with cardiac chest pain.[8]

The incidence of acute myocardial infarct with cocaine use is as high as 6% in adults.[9,10] However, the incidence with the use of methamphetamine and other illicit drugs is not reliably reported in the literature. Pediatric incidence of acute myocardial infarction due to cocaine use is unknown, although screening for illicit drugs in teenagers with chest pain should be considered.[11] The pathophysiology of chest pain associated with illicit drug use is commonly coronary artery vasospasm, which may or may not be associated with underlying atherosclerotic or congenital cardiac disease, having implications for treatment options. Cocaine acts as a sympathomimetic agent by blocking the reuptake of norepinephrine and dopamine at presynaptic adrenergic terminals, leading to excessive postsynaptic stimulation, thus increasing overall oxygen demand by the heart.[12] Cocaine also leads to premature coronary thrombus formation and is associated with early onset coronary artery disease and premature coronary artery plaque formation.[13]

Adult and pediatric patients are unreliable at self-reporting drug use.[14] Physiologic changes seen on physical examination include elevated systolic and diastolic blood pressures with increased mean arterial pressures compared with noncocaine users presenting with chest pain. Heart rate is increased due to sympathomimetic effects, and patients may exhibit tachypnea and diaphoresis.[10] Electrocardiographic findings consistent with acute coronary ischemia, arrhythmias, or pericarditis may be found, and serum cardiac enzymes may be elevated in patients with chest pain who use

cocaine or methamphetamine. Angiography frequently is normal, with no or minimal evidence of atherosclerotic cardiac artery disease.

Treatment of chest pain associated with ischemia induced by cocaine is similar to patients presenting with acute coronary syndrome, and should include antiplatelet drugs such as aspirin and clopidogrel. Beta-blockers should be avoided, as unopposed alpha-adrenergic stimulation can lead to increased coronary artery vasospasm and worsening cardiac circulation and ischemia. Benzodiazepines should be instituted as an early management strategy, because they decrease the central stimulation of cocaine, decrease actual chest pain, and can improve cardiovascular hemodynamics.[15] Nitroglycerine also has been shown to decrease coronary artery vasospasm in patients who have used cocaine and present with evidence of ischemia.[16] Angiography frequently is normal, with no or minimal evidence of atherosclerotic cardiac artery disease. Long-term, ongoing drug treatment may be required for these patients.

AORTIC DISSECTION

Aortic dissection is rare in the pediatric population, with estimates being less than 0.1% of chest pain cases. Aortic dissection, however, can be life threatening if not recognized early and treated aggressively. Aortic dissection can be associated with various pediatric conditions, including trauma, Marfan syndrome, Ehlers-Danloss syndrome, congenital bicuspid aortic valve, coarctation of the aorta, vasculitis, cocaine use, recent cardiac surgery, or aortic cannulation as seen with congenital heart disease.[17] Traumatic aortic dissection accounts for nearly 50% of cases of aortic dissection under the age of 21 years, and Marfan syndrome accounts for roughly 25% of cases.[18] In Marfan syndrome, aortic dissection is associated with aortic root dilatation, and the incidence is increased during pregnancy. Cocaine and other illicit drug use likely contribute to the increased incidence of aortic dissection through various mechanisms, most commonly unchecked hypertension.

An aortic root diameter of greater than 6 cm in adults is associated with the greatest risk of rupture, and intervention is recommended for aortic root diameters of greater than 5.5 cm. In trauma, however, there is usually no dilatation, but rather the force of the impact may disrupt the aortic root.

The pain typically is described as a sudden onset, sharp, “tearing” or “ripping” pain that may be anterior in the chest or radiating to the back. Patients also may present with abdominal pain, depending on the extent of the dissection, and more rarely with syncope or extremity and organ blood flow disruption from the extent of the dissection.[19]

In someone with a recent history of surgery, pneumonia, pulmonary embolism, or complications of surgery should be part of the differential for the cause of chest pain. For a patient with Marfan syndrome, it is important to also consider spontaneous pneumothorax as a cause of chest pain. Patients with congenital heart disease additionally may have disease-specific considerations, pericardial effusions, or infectious etiologies for chest pain.

The diagnostic gold standard for aortic dissection had been aortic angiography; however, in recent years computed tomography (CT), cardiovascular magnetic resonance imaging, and cardiac ultrasound all have been shown to be accurate and less invasive methods for the diagnosis of aortic dissection.[20] Chest radiography may show a widening of mediastinal structures in up to 85% of patients for those with traumatic aortic disruptions or high thoracic aortic dissections. Electrocardiographic findings may suggest left ventricular strain, hypertrophy, cardiac ischemia, nonspecific ST-T wave changes, or even an acute myocardial infarct pattern.[19]

Initial treatment involves controlling both blood pressure and heart rate to minimize the stress on the wall of the aorta while preparing for definitive operative repair. The goal values for adult systolic blood pressure are between 100 and 120 mm Hg, with a heart rate around 60 beats per minute. The goal values for vital signs should be appropriately age-adjusted in the pediatric population. Beta-blockers are the mainstay of therapy, as they also control blood pressure and heart rate, although peripheral vasodilators also can be helpful in decreasing blood pressure and left ventricular contractility forces. Many beta-blockers, such as labetalol, have extremely short half-lives. Sodium nitroprusside is commonly used in pediatric hypertensive crises, but should not be used as monotherapy in acute aortic dissections, because it can raise the peak maximal rate of pressure rise in the left ventricle during contraction, thus potentially expanding or rupturing an aortic dissection.[19]

For patients with underlying medical conditions such as Marfan syndrome, primary prevention with control of hypertension, aortic root dilatation monitoring, and early intervention is the best option. Definitive treatment of acute aneurysms requires prompt recognition, blood pressure and pulse control, and definitive surgical intervention. Despite monitoring and prompt evaluation, acute aortic dissections have a very high mortality rate.

CHEST TUMORS/MALIGNANCY

Primary tumors arising from mediastinal structures are rare, and more commonly occur in the adult population. The average age of presentation for patients with mediastinal tumors is 30 years old.[21,22] The ratio of males to females is roughly 2.8:1. Chest tumors in the pediatric population are likely to be neuroblastoma, lymphoma, or primary neurectodermal tumors (PNET).[22] The location of the tumor (anterior, middle, or posterior) within the mediastinum can suggest possible etiologies. Lymphoma, teratomas, thymomas, and thyroid tumors are more likely in the anterior and middle mediastinum, and neuroblastoma, neurofibroma, sarcoma, and germ cell tumors are more common in the posterior mediastinum.[23]

Patients may present with cough, wheezing, stridor, and anterior chest pain that is characterized by a deep sensation. Chest pain is found in half of all patients who present with a new diagnosis of mediastinal tumor, and it is usually not associated with activity or movement. Respiratory symptoms may be related to activity, and cough is found in over 80% of patients. Less commonly, supraclavicular lymphadenopathy, pleural effusions, symptoms consistent with superior vena cava syndrome, or respiratory distress or failure may be the presenting symptoms.[22,23]

Identification of the mass may be made by chest radiography or CT, but a biopsy is needed to confirm mass type, malignancy status, and treatment options. Fine needle aspiration, percutaneous lymph node biopsy, serum tumor markers, and mediastinoscopy or thoracostomy are all methods of obtaining tissue samples when chest masses have been identified. Individual therapy and outcome depend on the severity of the presentation and etiology of the tumor.

Chest pain with leukemia has been reported and may be due to mediastinal mass, mediastinal lymphadenopathy, focal bony destruction, pathologic fracture, pericarditis, pulmonary infarction from leukemic sludging, or coronary ischemia from direct infiltration of leukemic cells into the coronary arteries. In one case report, a 7-year-old with leukemia presented with vague, reproducible chest pain initially thought to be caused by costochondritis. He had persistent pain, but a rather unremarkable examination, and it is not clear what prompted the clinicians to obtain a complete blood count, which revealed the diagnosis.[24]

HERPES ZOSTER

Herpes zoster of the chest wall, or shingles, is a common cause of adult chest pain and is also well recognized in the pediatric population. The lesions are caused by reactivation of latent varicella zoster virus in the dorsal root or cranial nerve ganglion. It is seen with increasing frequency in the pediatric population among those who are immunosuppressed or immunocompromised, such as those with advanced HIV disease.[25] Herpes zoster occurs in healthy children who have had wild-type infection as well as children who have received the primary herpes zoster vaccination, although the incidence of herpes zoster is lower in vaccinated children. Primary vaccination does not prevent herpes zoster, and the isolate of varicella zoster causing the outbreak can be either wild type, vaccine strain, or mixed.

Patients present with pain that typically precedes rash formation, and the pain can vary in both character and intensity.[26] It often is described as burning, sharp, or aching, and is located unilaterally in a dermatomal pattern. Following the pain, an erythematous macular-turning vesicular rash characterized by crusting lesions can appear and follows the same dermatomal pattern as the pain. More so in children, fever may be associated with the presentation of herpes zoster. Treatment involves the use of antiviral medications such as acyclovir, famciclovir, and valacyclovir. Although this treatment has little impact on the acute course of zoster, it does decrease the incidence of postzoster neuralgia.[27] Postzoster neuralgia is pain associated with an outbreak that lasts for greater than 30 days after the onset of the initial rash, and this pain can be debilitating for patients.[28] Patients with lesions need to be placed in contact isolation for a minimum of 5 days after the onset of skin lesions, as contact with herpes zoster can transmit varicella to unvaccinated or immunocompromised patients. Herpes zoster is most commonly transmitted by direct contact with secretions from the skin lesions, but risk of transmission drops after the lesions have crusted over.[29]

SICKLE CELL DISEASE AND CHEST PAIN

Patients with sickle cell disease (SCD) are subject to a number of circumstances in which they are likely to experience chest pain as a consequence of acute worsening of their chronic hemolytic anemia. Vasoocclusive pain crises occur when sickled red blood cells (RBCs) obstruct blood flow and cause tissue ischemia. While isolated chest pain is not commonly described, individual variation in the location and severity of painful crises makes this a possible cause of chest pain for these patients. Infectious conditions, such as septic arthritis and osteomyelitis, often from encapsulated organisms, are more common in patients with SCD. Isolated costochondral, rib, or sternum involvement is rare, but the diagnosis should be entertained, especially in the setting of fever. Viral infections, particularly parvovirus B19, may result in marrow suppression, leading to hemoglobin drops that precipitate pain crises or congestive heart failure in the patient with SCD. Less commonly, an aplastic crisis may result from the folate deficiency common in SCD. Similar drops in hemoglobin may be seen when abnormal sickle shaped-cells are sequestered in the spleen during a splenic sequestration crisis. Ischemia or referred pain from the rapidly expanding spleen may lead to left-sided pain. Again, isolated chest pain in each of these settings would not be common, but only consideration will identify them.

The most serious and likely cause of isolated chest pain in the patient with SCD is acute chest syndrome. Patients present with a history of pain, frequently with fever, cough or some evidence of respiratory distress. The specific constellation of symptoms at presentation is highly variable and depends on age. Wheezing, cough, and

fever are more common in the young, and pain and dyspnea are more often seen in adult patients. Less common symptoms include abdominal pain, rib or sternal pain, neurologic dysfunction, cyanosis, or heart failure. Because no single sign or symptom is pathognomonic for acute chest syndrome, and almost half of patients may present for a different reason such as fever, hypoxia, or pain elsewhere in the body, a high degree of suspicion is necessary to ensure prompt diagnosis of acute chest syndrome. The mean age at first episode of acute chest syndrome is 14 years. Even in the younger pediatric age range, a history of acute chest syndrome is common, and recurrences are frequent. Patients older than 20 years of age diagnosed with acute chest syndrome have a more severe course than those who are younger, including longer hospitalization and more interventions while hospitalized.

The pathophysiology of acute chest syndrome is not well understood. Less than half of cases are found to have a precipitating factor such as infarction, infection, pulmonary fat embolism, or asthma exacerbation.[30,31] When an infectious cause is identified, the most common etiologies are *Chlamydia*, *Mycoplasma*, or viral. Bacterial causes are less commonly identified as precipitating acute chest syndrome. When identified, the bacteria most often associated with acute chest syndrome include *Staphylococcus*, *Streptococcus*, or *Haemophilus influenza.* Infrequent cases have been attributed to *Legionella* and mycobacterium species. Bacterial etiologies are usually identified in fatal cases of acute chest syndrome. Testing may reveal progression of symptoms, including worsening hypoxia, decreasing hemoglobin values, and progressive multilobar chest radiograph infiltrates. Elevation of the white blood cell count is common.

The general management of painful crises in SCD includes cautious and judicious hydration with isotonic solution to ensure euvolemia. Dehydration can contribute to RBC sickling, and overhydration may lead to pulmonary edema and congestive heart failure. Pain should be treated aggressively, most often with acetaminophen (with or without oxycodone), nonsteroidal anti-inflammatory drugs (NSAIDs) (such as ketorolac, if no prior NSAIDs taken), and potentially narcotics (morphine sulfate is most commonly used in the pediatric population). Controlling pain in patients with sickle cell disease prevents the respiratory splinting that is thought to contribute to progression of the vaso-occlusive crisis to acute chest syndrome. Careful monitoring for hypoventilation caused by respiratory drive suppression is important. Because hypoxia is common during acute chest syndrome and can lead to further RBC sickling, oxygen is commonly administered to patients with oxygen saturations less than 90%. The specific treatment of acute chest syndrome includes early consideration of packed red cell transfusion and possible exchange transfusion. Antibiotics (broad-spectrum, including a macrolide) should be administered. Bronchodilators may be appropriate in selected cases.[32]

BREAST SWELLING, MASSES (PREGNANCY, PHYSIOLOGIC)

Patients with breast disorders may present with the complaint of chest pain. Diseases of the breast are uncommon among pediatric patients, but may occur at any age. Boys more frequently complain of unilateral breast swelling and tenderness, especially at the onset of puberty. The breast nodule may be tender to palpation but in the absence of discharge, overlying skin changes, or lymphadenopathy, this may be considered benign and managed with reassurance only. Breast tenderness in teenage females during the menstrual cycle is common. Up to 50% of women experience fibrocystic disease, and pain associated with this condition commonly increases before onset of the menstrual cycle and resolves with the onset of menstruation.[33] Some teenage girls who present with the complaint of breast tenderness are found to be pregnant.

The clinician should consider this possibility when evaluating teenage girls with chest pain or breast tenderness.

Breast lumps in women are most commonly fibroadenomas. Hamartomas and other benign breast tumors are rare but present similarly as a solitary, mobile, and sometimes tender mass. Malignancy potential of both fibroadenomas and hamartomas is extremely low, particularly in younger patients, although patients and their families may find confirmation of nonmalignancy via excision biopsy to be reassuring.[34] Infection and localized abscess of the breast may be seen in the setting of body piercing.[35]

PLEURODYNIA

Pleurodynia is an uncommon cause of severe chest or upper abdominal pain. Also known as Bornholm disease, historically pleurodynia was an epidemic febrile disease of older children and young adults. Today both outbreaks and sporadic cases are occasionally seen. Several coxsackieviruses and echoviruses have been associated with pleurodynia; case reports with other viruses, such as herpes simplex, have been reported as well.[36] Although the pain intensity is variable, abrupt onset of excruciating pain spasms are typical and often lead to profuse sweating and pallor. The pain is often described as stabbing in quality and is thought to be muscular in origin. Its duration may be from minutes to hours (15 to 30 minutes most common), and it is exacerbated by sneezing, coughing, or deep inspiration. Associated symptoms are similar to enteroviral infections and include anorexia, vomiting, sore throat, and headache. While the symptoms are usually short lived (1 to 2 days), the painful episodes are frequently biphasic (rarely may occur more frequently), with repeat pain and fever occurring several days after the initial symptoms have resolved.

Physical examination may reveal tenderness at the site of pain, but the presence of erythema and swelling are uncommon. A pleural friction rub may be auscultated during pain episodes, but it is frequently absent when the pain has diminished. Rapid, shallow, grunting respirations may suggest pneumonia or pleural inflammation. Diagnosis is suspected by clinical history and consideration of season, viral presence in the community, and incubation period. Diagnostic confirmation, when needed, is obtained by polymerase chain reaction (PCR) of infected fluids (sputum, blood, stool, or pleural or spinal fluid), with culture being possible but less helpful clinically because of the time required for growth. Ancillary data most commonly demonstrate an elevated white blood count (frequently caused by polymorphonuclear neutrophils and band forms), an elevated erythrocyte sedimentation rate and C-reactive protein, and a normal chest radiograph.

Treatment is typically supportive, with analgesia for pain and antipyretics for fever. Reassurance and teaching of prevention measures related to the natural history of prolonged excretion in the stool are required to decrease spread of the disease. For severe cases in the young or immunocompromised patient, consideration of immunoglobulin therapy may be warranted. Commercially available immunoglobulin preparations have been shown to contain antibodies to most enteroviruses, and although published data do not support a clear benefit, this may be a useful adjunct to consider.[37]

OTHER CAUSES

Severe chest pain warrants consideration of several other rare entities that may mimic the entities already discussed. *Cervical arthritis* can produce radicular pain that is likely to be focal, severe, and described as sharp.[38] *Cysts of the spinal cord* are uncommon but can cause precordial pain that may mimic pleurodynia.[39] Intraspinal cysts may present with isolated intermittent pain that is difficult to diagnose; chronicity,

increasing severity, and neurologic findings such as paresthesia and intermittent spastic weakness may give additional clues to this condition. Symptoms and diagnosis are generally established in the first decade of life. Complete excision is the definitive treatment, but drainage may provide relief of pain. Furthermore, *osteomyelitis of the rib* has been reported as a very rare cause of chest pain.[40] The patient with this rare infection is likely to have fever and reproducible chest pain.

Chest pain is rarely caused by a sprain disorder known as *slipping rib syndrome*. This is caused by trauma to the costal cartilages of the 8th, 9th, and 10th ribs that do not attach to the sternum. Children with slipping rib syndrome complain of pain under the ribs or in the upper abdominal quadrants.[41] They also may report hearing a clicking or popping sound when they lift objects, flex the trunk, or walk. It is believed the pain is caused when one of the ribs hooks under the rib above it and irritates the intercostal nerves. The pain can be duplicated and the syndrome confirmed by performing the hooking maneuver, whereby the affected rib margin is grasped and then pulled anteriorly. The only definitive management is surgical, although most patients are treated satisfactorily with nonopioid analgesics.[41]

Tietze syndrome is another rare condition of unknown etiology that causes sharp or stabbing sternal chest pain. Physical examination of children with this condition may reveal tender, spindle-shaped swelling at the sternochondral junctions, which differentiates this condition from costochondritis. This syndrome is diagnosed clinically and can last for months. Suggested treatments include reassurance, local application of heat, and nonsteroidal anti-inflammatory agents.[42]

Finally, a recent case of a teenager with sudden, severe, midsternal chest pain after multiple episodes of forceful vomiting offered a surprising diagnosis of *Boerhaave syndrome*.[43] The patient's emesis had some brownish material, and he had tachycardia on presentation, but no subcutaneous emphysema was appreciated. A chest radiograph revealed a pneumomediastinum extending into the soft tissues of the neck. A ruptured esophagus was suspected, and a contrast esophagogram confirmed this. Boerhaave syndrome, spontaneous nontraumatic rupture of the esophagus, is different than Mallory-Weiss syndrome, as the former involves transmural rupture, and the latter involves longitudinal tears on the lower esophageal mucosa. A history of vomiting, chest pain, and the finding of subcutaneous emphysema should raise the suspicion of Boerhaave syndrome.

Early recognition and management of the both the common and more obscure causes of pediatric chest pain, which is most often not of cardiac origin, may help to avoid unnecessary referral and can alleviate the concern and stress families and patients experience when dealing with chest pain.

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